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**Mood Disorders and the Brain:
Depression, Melancholia, and the Historiography of Psychiatry**

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Introduction: ‘Mood Disorders’ as Historical Problem

Despite the increasingly widespread availability of psychotropics believed to restore biochemical equilibrium in the brains of persons diagnosed with mood disorders,ⁱ the number of people suffering from such medical conditions appears to be increasing. According to The Royal College of Psychiatrists, ‘by 2020 it is estimated that depression will be the second most common disabling condition in the world’, a figure it derives from the World Health Organization.ⁱⁱ Depression is, it seems, rapidly becoming a global threat. In a trend that is mirrored in much of the West, the number of prescriptions dispensed for antidepressants in the UK has doubled in the last decade and is continuing to rise. The need for a critical perspective on mood disorders is growing.

At the start of the millennium, Mikkel Borch-Jacobsen lamented the ‘epistemic timidity’ of historians of psychiatry, when they ‘are in fact in a position of saying something of capital importance about the subject of mental illness.’ⁱⁱⁱ Reiterating this, I want to suggest that historians of psychiatry are best suited to provide a critical perspective on mood disorders and that they must continue to broaden and deepen their research field in order to do so.

We can begin by considering how the concept of ‘mood disorders’ emerged in the early decades of the nineteenth century. Such a history must also be a history of the emotions and of the sciences that investigate these. Moreover, as with any history concerned with disease concepts,^{iv} historians of psychiatry must pay close attention to nosological texts in tracing how psychiatric categories were created in relation to clinical practices and to other realms of medical science. Finally, our inquiry must incorporate contemporaneous developments in experimental neurophysiology that nineteenth-century psychiatrists, particularly in Germany and Britain, drew extensively upon to explain how emotions functioned and could become diseased.

Twenty-first century psychiatrists, psychologists, and neuroscientists are fond of looking to the nineteenth century in support of claims for continuity and universality of present knowledge about the emotions and their disorders.^v Historians, too, have attempted to establish parallels between today's clinical depression and nineteenth-century conditions such as melancholia and neurasthenia.^{vi} Such claims should not, however, be taken for granted, nor are the presumed links inevitable.

This paper provides a short narrative on how melancholia was reconceptualised by an influential German psychiatrist, Wilhelm Griesinger (1817–68), in the middle of the nineteenth century. He drew on neurophysiological work on the sensory–motor reflex, applying the reflexive model to the realm of ideas and emotions in order to situate the recently created category, ‘affective insanity’, within a biomedical framework. Through the metaphor of reflexive action, the emotions could be explained as the result of a potentially involuntary mental process with the ability to become disordered. Nineteenth-century melancholia, conceptualised within this framework, was specific to a particular historical moment – it had its own regime of somatisation and biologisation, and its own aetiology and trajectory. It follows that if the historical specificity of nineteenth-century biomedical melancholia can be demonstrated, depression as a universal condition must equally become open to re-interpretation and be subjected to closer historical investigation.

However, the story presented not only highlights historical difference: what also emerges is a *continuity of language* which facilitates the idea of disordered emotions as scientifically sound in the twenty-first century. Griesinger's metaphor has survived to the present, providing a linguistic template that allows for ‘mood disorders’ to exist as a plausible and meaningful medical concept.

The Emergence of Disordered Emotions

German Berrios has suggested that:

[Up] to the Napoleonic Wars, melancholia was but a rag-bag of insanity states whose only common denominator was the presence of a few (as opposed to many) delusions. In practice, therefore, it is highly likely that it included cases of schizophrenia. Sadness and low affect (which were no doubt present in some cases) were not considered as defining symptoms. Indeed, states of non-psychotic depression, of the type that nowadays would be classified as DSM IV ‘Major Depressive Episode’ would not have been called ‘melancholia’ at all. During the eighteenth century these states were classified as ‘vapours’, ‘spleen’, or ‘hypochondria’,

i.e. what Cullen called ‘neuroses’, and Sydenham and Willis, the previous century, had called ‘nervous disorders’.^{vii}

‘Retrospective diagnosis’ is a practice from which historians of medicine often seek to distance themselves, yet few have questioned the claim that clinical depression has always been part of the human condition, albeit under a variety of names. It is tempting to take for granted that as humans we have been subject to and are capable of the same kinds of emotions throughout our history and that that, in fact, is a large part of what makes us human. However, historians of the emotions have suggested that emotional repertoires change over time and between societies.^{viii} Moreover, as Thomas Dixon has shown, ‘emotions’ as a concept has its own history. It emerged within the context of modern psychology, gradually replacing older terms such as passions, appetites, and sentiments that had belonged to and made sense within a world-view where Christianity was the dominant source of knowledge. As ways of understanding the human mind and body were profoundly transformed within a modern scientific framework, ‘the emotions’ as an all-compassing category and process that was at once physiological and psychological – and not necessarily subject to volition – soon eclipsed its spiritual predecessors in medico–scientific literature.^{ix}

When investigating how mental disease categories emerge and function, historians of psychiatry cannot, however, confine themselves to considering external symptoms – in this case emotional expressions. How these conditions are internally explained is equally central to their history. In this way, depression, as presently understood, has a descriptive psychopathological component, based on ‘symptoms’ patients express; but an equally important, and defining, part of this condition is that it is understood as a biochemical imbalance, which can potentially be corrected with antidepressants. Following from this, one cannot simply equate nineteenth-century melancholia and twentieth- and twenty-first-century depression either with each other or with the nervous disorders of the seventeenth century, or the neuroses of the eighteenth.

With the rise of a new brand of European psychiatry in the middle of the century melancholia was reconfigured as an emotional disorder with a neurophysiological basis. Wilhelm Griesinger was one of the early figures central to bringing about this development. His system of classification awarded a comparatively prominent place to melancholia, which he viewed as the most treatable form of madness. What has been curiously overlooked, however, is how Griesinger came to arrive at his conceptualisation of melancholia as a biomedical disorder of the emotions. A closer look at his theory of disordered emotions sheds

light on how the concept of mood disorders became possible and plausible. It also highlights differences rather than uniformity where melancholia and clinical depression are concerned, suggesting that these are two distinct disease concepts whose historical trajectories demand close and critical inquiry, and which it is the business of historians of psychiatry to trace and explicate.

In constructing his psychiatric nosology, Griesinger drew to an extent on the work of earlier mental scientists, particularly Esquirol and Guislain, as well as on his own clinical experience. However, in arriving at an internal, biomedical model for mental disease, he primarily found his inspiration in experimental neurophysiology, particularly the work of Johannes Müller. The latter's concept of mental reflex was at the heart of the model Griesinger put forward, proving the foundation for his understanding of how the emotions could become diseased.

With experiments carried out on living animals by nineteenth-century physiologists the concept of reflex action was dressed in the language of modern empirical science.^x Müller built on the sensory–motor reflex arc developed by Marshall Hall early in the century. Hall, however, rejected the idea that the brain might be involved in reflex action, marking the medulla as the final destination point for external stimuli. Müller disagreed on this point, since his experiments led him to believe that there must be cerebral involvement in the reaction that occurred. He proposed a reflex occurring in the mind, a model which was soon extended beyond a model for external sensation triggering bodily movement into the realm of ideas.^{xi} This emerging neurophysiological mental reflex would provide an explanatory framework for how the emotions were produced.

In an 1843 article published two years prior to the first edition of his influential textbook, Griesinger sought to explain mental, and particularly emotional, functioning through the reflex concept. The paper 'Ueber psychische Reflexactionen'^{xii} has received scant attention by historians,^{xiii} despite laying the foundation for Griesinger's vastly influential psychiatric nosology.

Griesinger's Pathology of Ideas and Emotions:

From Mental Reflex to Cerebral Irritation

Griesinger followed Müller in suggesting that the brain is involved in reflex action, arguing in addition to this that sensation could pass through the brain, triggering reaction, *without alerting consciousness*. Moreover, such transmission of unconscious sensation is conducted more smoothly and efficiently than that involving the will. All impressions, however,

whether registered in the awareness or not, are collected in the brain, where they are transformed into ideas, or mental images [*Vorstellungen*]:

Some sensations do not reach the consciousness, and others do. Both kinds of impressions form the totality of all exciting factors [*Erregungen*] of the central organ [the brain]; the entire content of the sensory site of the latter is equal to the content of all centripetal impressions.... The central organ does not hold these impressions in individual fibres, but produces from all of them, continuously, a script [*Facit*] containing the summary of all excitations. When this state is seemingly calm it regulates and upkeeps the tone [of the brain].... The centripetal impressions from the immediate site (the mental organ [*Sinnesorgane*]), and from the remote regions (from the spinal marrow outwards) are dispersed in the brain, where they undergo a further change and become the source of *Vorstellungen*.^{xiv}

The metaphor of a *Facit*, being the summary of all (external and internal) sensory impressions, allows Griesinger to describe how the mind operates. The *Facit* contains all mental information, some of which has passed through the consciousness, and some that has been stored without triggering awareness. All these impressions are capable of reacting upon one another, and in doing so they have the ability to synthesise and create new ideas (*Vorstellungen*). Such *Vorstellungen* are wholly endogenous, and may be utterly unrelated to any external stimuli. Moreover, ideas are kept in ‘mental storages’ [*geistigen Vorraths*], and when new impressions reach the brain from outside, these are prone to react with the previously stored images to produce new ones.^{xv}

It is this ability of the brain to react both to external and internal stimuli, and to produce from any combination of these entirely new impressions and mental images, which is the source of mental disease. When an increasing number of negative impressions are stored and these react both with further external irritants and with each other internally, the brain is subjected to repeated ‘irritation’ [*Reize*]. Eventually, the *process* of mental reaction – the reflexive action – becomes distorted, and subsequently diseased. The brain begins to produce morbid reactions, such as feelings of displeasure [*Unlust*], in response to factors that would not trigger such reactions in a healthy mind.^{xvi}

There are, then, two metaphors at work here which together make up Griesinger’s model for the emotions: a script containing mental images, or ideas, and the ability of these ideas to react upon one another through a process similar to sensory–motor reflex action. The

mental reflex as expounded by Griesinger was subsequently taken up by other European psychiatrists, such as Henry Maudsley in Britain, and Richard von Krafft-Ebing in Germany,^{xvii} and came to constitute a central element in the biomedical psychiatry of the mid-to-late nineteenth century. It provided the necessary mechanism by which milder forms of insanity – disorders of affect – could be explained. These were conditions which did not normally turn up lesions of the brain during psychiatric autopsies,^{xviii} thus it offered a way of explaining milder forms of insanity within a medico–scientific – in this case neurophysiological – framework.

Conclusion

For Griesinger, the starting point for all forms of insanity was the ‘states of mental depression’ which included hypochondria, and simple and delusional melancholia.^{xix} These formed the first pathological stage to be induced by morbid reflex action resulting from persistent irritation of the brain. If left untreated, melancholia could and often did deteriorate into mania and later dementia, an idea that can be found in the writings of a number of prominent British mental scientists in the second half of the nineteenth century.

Nineteenth-century biomedical melancholia bears little resemblance to today’s depression. The former is a disorder of the emotions where the intellect remains largely unaffected in the early stages of the disease. External and internal stimuli react upon the brain, cause irritation, and produce morbid emotional reactions. This leads to low mood, introspection, and despair, often accompanied by intense feelings of guilt and thoughts of suicide. If patients are not treated quickly, their mental state deteriorates, and they start to experience delusions, often of a religious nature. This is the progression from simple to delusional melancholia that, if left untreated, may eventually progress into mania and dementia.^{xx}

Clinical depression is a mood disorder characterised by depressed mood, inertia, appetite changes, lethargy, indifference, difficulty thinking and concentrating, feelings of guilt and worthlessness, and thoughts of suicide.^{xxi} The condition strikes women to a much larger extent than men.^{xxii} External factors and internal brain chemistry are seen to work in conjunction to produce morbid symptoms. Internally, the illness is linked to deficiencies of a number of neurotransmitters, particularly serotonin and dopamine. The drugs currently most favoured to treat this condition are believed to regulate postsynaptic re-uptake of serotonin, thus improving and stabilising mood.^{xxiii}

However, while we are dealing with two separate disease concepts, both of which must be subjected to closer historical scrutiny, they are made meaningful through the same metaphor. The idea that emotional reactions occur reflexively and involuntarily in response to internal and external stimuli persists in the present and continues to make possible and plausible the concept mood disorders.

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ⁱ As presently defined in Anglo-American psychiatry, the category ‘mood disorders’ incorporates various depressive disorders, as well as bipolar disorder. See, for example, *Diagnostic and Statistics Manual of Mental Disorders (DSM-IV-TR)* (Arlington, VA: American Psychiatric Association, 2000), 345–7.

ⁱⁱ ‘A joint statement by the Royal College of General Practitioners (RCGP) and the Royal College of Psychiatrists (RCPsych)’, ‘Patients and Antidepressants’, Royal College of Psychiatrists (March 2008),

<http://www.rcpsych.ac.uk/mentalhealthinfoforall/problems/depression/patientsandantidepressants.aspx>, accessed 2 February 2011.

ⁱⁱⁱ Mikkel Borch-Jacobsen, ‘Making Psychiatric History: Madness as Folie à Plusieurs’, *History of the Human Sciences*, 14, 2 (2001), 23–4.

^{iv} I broadly follow Adrian Wilson’s approach in this regard: Adrian Wilson, ‘On the History of Disease-Concepts: The Case of Pleurisy’, *History of Science*, 38, 3 (2000), 271–319.

^v Examples abound, such as Rebecca Fox-Spencer and Alan Young, *A Simple Guide to Depression* (Long Hanborough: CFS Medical Communications, 2005), 34–5; Michael Alan Taylor and Max Fink, *Melancholia: The Diagnosis, Pathophysiology, and Treatment of Depressive Illness* (Cambridge: Cambridge University Press, 2006), 1–7; Allan V. Horowitz and Jerome C. Wakefield, *The Loss of Sadness: How Psychiatry Transformed Normal Sorrow into Depressive Disorder* (Oxford: Oxford University Press, 2007), 53–4; 61–71; Dan J. Stein, David J. Kupfer and Alan F. Schatzberg (eds), *The American Psychiatric Publishing Textbook of Mood Disorders* (Washington, DC: American Psychiatric Publishing, 2006). This claim for continuity is presented in unusual detail in a collaborative project between an historian and a psychiatrist: Conrad M. Swartz and Edward Shorter, *Psychotic Depression* (Cambridge: Cambridge University Press, 2007). It should be noted that the

'discovery' of depression is usually traced further back than the 1800s, often to Antiquity. There is, however, insufficient space here to address the many problems arising from attempts to link modern depression to a multitude of pre- and early modern afflictions.

^{vi} For example, Stanley W. Jackson, *Melancholia and Depression: From Hippocratic Times to Modern Times* (New Haven: Yale University Press, 1986); Judith Misbach and Henderikus Stam, 'Medicalizing Melancholia: Exploring Profiles of Psychiatric Professionalization', *Journal of the History of the Behavioural Sciences*, 42, 1 (Winter 2006), 41–59; Janet Oppenheim, *'Shattered Nerves': Doctors, Patients, and Depression in Victorian England* (Oxford: Oxford University Press, 1991).

^{vii} German E. Berrios, *The History of Mental Symptoms: Descriptive Psychopathology since the Nineteenth Century* (Cambridge: Cambridge University Press, 1996), 299.

^{viii} See Karin Johannisson, *Melankoliska rum: om ångest, leda och sårbarhet i förfluten tid och nutid* (Stockholm: Albert Bonniers, 2009); Barbara H. Rosenwein, 'Worrying about Emotions in History', *American Historical Review*, 107 (June 2002), 821–45.

^{ix} Thomas Dixon, *From Passions to Emotions: The Creation of a Secular Psychological Category* (Cambridge: Cambridge University Press, 2003). See also Roger Smith, 'The History of Psychological Categories', *Studies in History and Philosophy of Science, Part C: Studies in History and Philosophy of Biological and Biomedical Sciences*, 36, 1 (March 2005), 55–94.

^x For a history of the reflex in modern science see Georges Canguilhem, *The Vital Rationalist: Selected Writings from Georges Canguilhem* (New York: Zone Books, 1994): Ch. 8, 'The Concept of Reflex'. For a different, but equally brilliant historical approach to reflex action, see Jean Starobinski, *Action and Reaction: The Life and Adventures of a Couple* (New York: Zone Books, 2003).

^{xi} Johannes Müller, *Elements of Physiology, Vol. I* (London: Taylor and Walton, 1840), 765–95, and *Vol. II: The Physiology of the Senses, Voice, and Muscular Motion, with the Mental Faculties* (London: Taylor and Walton, 1848), 1354–64.

^{xii} Wilhelm Griesinger, 'Ueber psychische Reflexactionen: Mit einem Blick auf das Wesen der psychischen Krankheiten', *Gesammelte Abhandlungen, Ersters Band: Psychiatrische Abhandlungen* (Amsterdam: E.J. Bonnet, 1968).

^{xiii} A important exception being: Edwin Clarke and L.S. Jacyna, *Nineteenth-Century Origins of Neuroscientific Concepts* (Berkeley, CA: University of California Press, 1987).

^{xiv} Griesinger, *op. cit.* (note 12), 11–12. [whose translation?]

^{xv} *Ibid.*, 24.

^{xvi} *Ibid.*, 24–5; see also Wilhelm Griesinger, *Mental Pathology and Therapeutics*, 2nd edn (New York: William Wood & Co, 1882), 23–4.

^{xvii} Henry Maudsley, *The Physiology and Pathology of Mind* (London: Macmillan, 1867) and 'The Cerebral Cortex and Its Work', *Mind*, 58 (1890), 161–90; Richard von Krafft-Ebing, *Melancholie:*

Eine Klinische Studie (Erlangen: Ferdinand Enke, 1874), 1–6, 9–11. For instance, William Sankey used the model as early as 1863 to explain the aetiology of melancholia, and a similar explanation through the mental reflex was expressed by Thomas Clouston in the 1890s. Thomas S. Clouston, *Clinical Lectures on Mental Diseases*, 5th edn (London: J & A Churchill, 1898), 14–15, 27–30; W.H.O. Sankey, ‘On Melancholia’, *Journal of Mental Science*, 9 (1863), 193–4.

^{xviii} Griesinger himself addressed this problem in his textbook: Griesinger, *Mental Pathology*, *op. cit.* (note 16), 4–5, 409–12. For a discussion on the emergence of psychiatric autopsies, see Eric J. Engstrom, *Clinical Psychiatry in Imperial Germany: A History of Psychiatric Practice* (Ithaca: Cornell University Press, 2004), 96–8.

^{xix} Griesinger, *Mental Pathology*, *op. cit.* (note 16), 209–72.

^{xx} See, for example, the works referenced in *op. cit.* (note 18) above.

^{xxi} *DSM-IV-TR*, 349.

^{xxii} Danuta Wasserman, *Depression: The Facts* (Oxford: Oxford University Press, 2006), 63; *DSM-IV-TR*, 372; Fox-Spencer and Young, *op. cit.* (note 5), 30.

^{xxiii} Fox-Spencer and Young, *ibid.*, 14, 17, 72–5; Wasserman, *ibid.*, 230–6.